Editorial Commentary: Trochlear Dysplasia: Can We Change its Natural History or Degenerative Prognosis?

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Abstract: Trochlear dysplasia may be asymptomatic and benign, or could engender patellar instability and degenerative arthritis. Autologous chondrocyte implantation is demonstrating promising outcomes for the treatment of patellofemoral cartilage lesions, but may not suffice for knees with underlying mechanical anomalies as trochlear dysplasia, where adjuvant trochleoplasty or tibial tubercle osteotomy may be required to prevent patellofemoral instability and to protect the graft from wear and damage. Rigorous radiographic assessment is important to discern the type of dysplasia, notably the presence of a potentially pathogenic supra-trochlear spur. Trochleoplasty or other realignment procedures such as tibial tubercle osteotomy should be considered where necessary to correct underlying trochlear deformities and thereby avoid iatrogenic complications or failure.

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Trochlear dysplasia is a condition, not a disorder. As such, it may be asymptomatic and benign, although it is often associated with patellofemoral pain or instability and could lead to degenerative arthritis. The ambivalence of trochlear dysplasia is evident from the number of bilateral cases that we treat, where one knee develops symptoms following patellar dislocation or injury whereas the other knee remains nonpathologic for decades thereafter. The remarkable study of Mestriner, Ackermann, Morlin-Ambra, Franciozi, Faloppa, and Gomoll,[1] entitled "Trochlear Dysplasia Does Not Affect the Outcomes of Patellofemoral Autologous Chondrocyte Implantation," compares mid-term outcomes of cartilage repair by autologous chondrocyte implantation (ACI) at the patellofemoral joint (PFJ) in 2 matched cohorts of knees with normal (n = 23) and dysplastic (n = 23) trochleae. The authors are to be applauded for their meticulous follow-up of patients with such rare and complex indications and for applying sophisticated matching algorithms to minimize confounding variables.

Patellofemoral cartilage lesions have 2 root causes. The first is the underlying abnormalities that lead to kinematic malfunction and overstrain on different parts of the patella and trochlea. The second is macroscopic and/or microscopic trauma every time the patella dislocates, relocates, or subluxates. Cartilage repair is demonstrating promising outcomes for the treatment of patellofemoral cartilage lesions,[2] and while it may be effective to treat acute traumatic defects, it would be of limited benefit for chronic atraumatic defects with underlying mechanical causes. And while the role of trochlear dysplasia in the development and deterioration of cartilage defects remains unclear, the relationship between trochlear dysplasia and isolated patellofemoral arthritis is well-documented, especially for high-grade dysplasia, characterized by a supratrochlear spur.[3,4] In my experience, many surgeons fail to notice the supratrochlear spur that characterizes type B and type D dysplasia and that exacerbates abnormal patellar tracking beyond the simple "J sign" and initiates localized cartilage damage. The symptoms are further aggravated in knees with malaligned extensor mechanism or patella alta.

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The extent of anterior trochlear overhang and the presence of a supratrochlear spur can only be measured on true lateral radiographs with the posterior femoral condyles superimposed, which is mandatory in addition to standard skyline radiographs and slice imaging (computed tomography or magnetic resonance imaging) when assessing trochlear dysplasia. While Mestriner et al.[1] used both lateral radiography and magnetic resonance imaging to assess trochlear dysplasia using the Dejour classification, they regrouped knees according to Lippacher et al.[5] as low-grade (type A) and high-grade dysplasia (types B, C, and D). In my opinion, this regrouping is not appropriate in the context of patellofemoral cartilage lesions, because it does not sort knees according to the most important factor, which is the presence of a supratrochlear spur. I would recommend that future studies investigate whether outcomes of patellofemoral cartilage repair are associated with the presence and size of supratrochlear spur, rather than with qualitative types of dysplasia.

The take-home messages of Mestriner et al.[1] are that "abnormal trochlear anatomy should not be considered a contra-indication for cartilage repair in the PFJ using ACI." and that "anatomic correction of the trochlear dysplasia may not be required to achieve good clinical outcomes in patients with PFJ chondral defects." Both conclusions seemed counterintuitive to me at first, but after some thought, they sounded plausible and indeed logical for cases in which patellofemoral instability was either not present or had already been treated. Before further elaboration, it is worth noting that while anatomic correction of trochlear dysplasia is possible by procedures such as sulcus-deepening trochleoplasty,[6] there are several other treatments for patellofemoral pain and instability, including combinations of targeted physiotherapy, medial patellofemoral ligament reconstruction, lateral patellar facetectomy, and tibial tubercle osteotomy (TTO). The ideal treatment depends on the clinical symptoms as well as the grade of trochlear dysplasia, the understanding of which is of paramount importance to avoid iatrogenic complications or failure.

Several studies suggested that adjuvant TTO improves outcomes of cartilage repair in the PFJ, in knees with both normal or dysplastic trochleae, because it realigns the extensor mechanism such that pressures at the grafted surfaces are minimized.[7,8] In the series of Mestriner et al.,[1] concomitant TTO was performed in 74% of dysplastic knees and in 57% of nondysplastic knees, which suggests that patellar instability was treated where present and that the remaining knees had little or no symptoms thereof. In other words, the remaining 26% of dysplastic knees in which concomitant TTO was not performed probably had benign or asymptomatic trochlear dysplasia, whence the satisfactory results of isolated ACI. The prevalence of the benign or asymptomatic form of trochlear dysplasia is likely lower than that, considering that the authors included only 46 (24%) of their consecutive series of 190, as they excluded 49 that had concomitant tibiofemoral ACI; 42 that had concomitant/previous procedures and/or incomplete data; 33 that either had <2 years of follow-up, inadequate imaging, or patella alta; and 20 that could not be matched. It would be interesting to confirm whether the conclusions hold if the analyses were performed on the initial series of 190 knees, or on the subgroup of 141 knees that had isolated PFJ ACI, and whether outcomes differed for medial lesions versus lateral lesions.

The study of Mestriner et al.[1] revealed promising outcomes of cartilage repair in the PFJ and interesting findings regarding treatment of knees with trochlear dysplasia. It was particularly intriguing to note that while postoperative Knee Injury and Osteoarthritis Outcome Score subcomponents were nearly identical for knees with normal versus dysplastic trochleae, the baseline values were considerably better (except for Quality of Life and Sports/Recreation), which suggests that knees with trochlear dysplasia generally adapt to impaired patellofemoral tracking and have a good potential for recovery if treated appropriately. I therefore encourage the authors to pursue future assessment of their cohort to ascertain whether good outcomes are maintained for both normal and dysplastic knees in the long term and whether similar outcomes can be expected for patients who were excluded from the initial series. In the meantime, surgeons must beware of overinterpreting those findings and should carefully consider trochleoplasty or other mechanical adjustments where necessary, as correcting underlying trochlear deformities may be the only way to prevent recurrence of dislocation and instability and to protect the cartilage graft from wear and damage.

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